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CABA,  
CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB,  
DDFU, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, ...' ENTERED AT 15:42:38 ON  
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SEA FUCOSYLTRANSFERASE

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QUE FUCOSYLTRANSFERASE

L1

FILE 'CAPLUS, BIO, EMBASE, BIOTECHNO, MEDLINE, SEARCH' ENTERED AT  
15:44:01 ON 18 SEP 2002

L2

33 S L1 AND (VITRO GLYCOSYLATION) OR (VITRO FUCOSYLATION)

L3

6 DUP REM L2 (27 DUPLICATES REMOVED)

=> d 13 ibib ab 1-6

L3 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 1  
ACCESSION NUMBER: 1999:294276 CAPLUS  
DOCUMENT NUMBER: 131:100358  
TITLE: The Effect of O-Fucosylation on the First EGF-like  
Domain from Human Blood Coagulation Factor VII  
AUTHOR(S): Kao, Yung-Hsiang; Lee, Geoffrey F.; Wang, Yang;  
Starovasnik, Melissa A.; Kelley, Robert F.; Spellman,  
Michael W.; Lerner, Laura  
CORPORATE SOURCE: Departments of Analytical Chemistry Protein  
Engineering, Pharmacokinetics and Metabolism  
Genentech  
Inc., South San Francisco, CA, 94080, USA  
SOURCE: Biochemistry (1999), 38(22), 7097-7110  
CODEN: BICHAW; ISSN: 0006-2960  
PUBLISHER: American Chemical Society  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
AB The first epidermal growth factor-like domain (EGF-1) from blood  
coagulation factor VII (FVII) contains two unusual O-linked glycosylation  
sites at Ser-52 and Ser-60. We report here a detailed study of the  
effect  
of O-fucosylation at Ser-60 on the structure of FVII EGF-1, its  
Ca<sup>2+</sup>-binding affinity, and its interaction with tissue factor (TF). The  
in **vitro** fucosylation of the nonglycosylated FVII  
EGF-1 was achieved by using O-fucosyltransferase purified from Chinese  
hamster ovary cells. Distance and dihedral constraints derived from NMR  
data were used to det. the soln. structures of both nonglycosylated and  
fucosylated FVII EGF-1 in the presence of CaCl<sub>2</sub>. The overall structure  
of  
fucosylated FVII EGF-1 is very similar to the nonfucosylated form even  
for  
the residues near the fucosylation site. The Ca<sup>2+</sup> dissocn. consts. (K<sub>d</sub>)  
for the nonfucosylated and fucosylated FVII EGF-1 were found to be 16.4  
+- 1.8 and 8.6 +- 1.4 mM, resp. The FVII EGF-1 domain binds to the  
extracellular part of TF with a low affinity (K<sub>d</sub> approx. 0.6 mM) and  
the addn. of fucose appears to have no effect on this affinity. These  
results indicate that the FVII EGF-1 alone cannot form a tight complex  
with TF and suggest that the high binding affinity of FVIIa for TF  
requires cooperative interaction among the four domains in FVII with TF.  
Although the fucose has no significant effect on the interaction between  
TF and the individual FVII EGF-1 domain, it may affect the interaction of  
full-length FVIIa with TF by influencing its Ca<sup>2+</sup>-binding affinity.  
REFERENCE COUNT: 74 THERE ARE 74 CITED REFERENCES AVAILABLE FOR  
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FORMAT

L3 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 2  
ACCESSION NUMBER: 1999:581084 CAPLUS  
DOCUMENT NUMBER: 131:320873  
TITLE: The formation of the oncofetal J28 glycotope involves  
core-2 .beta.6-N-acetylglucosaminyltransferase and  
.alpha.3/4-fucosyltransferase activities  
AUTHOR(S): Panicot, Laurence; Mas, Eric; Pasqualini, Eric;  
Zerfaoui, Mourad; Lombardo, Dominique; Sadoulet,  
Marie-Odile; El Battari, Assou

CORPORATE SOURCE: INSERM U 260, Unite de Recherche de Physiopathologie  
des Regulations Hormono-Nutritionnelles, Faculte de  
Medecine-Timone, Marseille, 13385, Fr.  
SOURCE: Glycobiology (1999), 9(9), 935-946  
CODEN: GLYCE3; ISSN: 0959-6658  
PUBLISHER: Oxford University Press  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB The feto-acinar pancreatic protein or FAPP, the oncofetal glycoisoform of  
bile salt-dependent lipase (BSDL), is characterized by the presence of  
the

J28 glycotope recognized by mAbJ28. This fucosylated epitope is carried  
out by the O-linked glycans of the C-terminal mucin-like region of BSDL.  
This glycotope is expressed by human tumoral pancreatic tissues and by  
human pancreatic tumoral cell lines such as SOJ-6 and BxPC-3 cells.  
However, it is not expressed by the normal human pancreatic tissues and  
by

MiaPaCa-2 and Panc-1 cells. Due to the presence of many putative sites  
for O-glycosylation on FAPP and BSDL, the structure of the J28 glycotope  
cannot be attained by classical phys. methods. In the first part of the  
present study, we have detd. which glycosyltransferases were differently  
expressed in pancreatic tumoral cell lines compared to normal tissues,  
focusing in part on fucosyltransferases (Fuc-T) and core-2  
.beta.6-N-acetylglucosaminyltransferase (Core2GlcNAc-T). The data  
suggested that .alpha.2-Fuc-T activity was decreased in the 4 cell lines  
tested (SOJ-6, BxPC-3, MiaPaCa-2, and Panc-1). The .alpha.(1-3) and  
.alpha.(1-4) fucosylations were decreased in tumor cells that do not  
express the J28 glycotope, whereas .alpha.4-Fuc-T and Core2GlcNAc-T  
activities were significantly increased in SOJ-6 cells which best  
expressed the J28 glycotope. Therefore, we wished to gain information  
about glycosyltransferases involved in the building of this structure by  
transfecting the cDNA encoding the mucin-like region of BSDL in CHO-K1  
also expressing Core2GlcNAc-T and/or FUT3 and/or FUT7 activities. These  
CHO-K1 cells have been previously transfected with the cDNA encoding  
Core2GlcNAc-T and/or FUT3 and/or FUT7. Data indicated that the

C-terminal

peptide of BSDL (Cter) produced by those cells did not carry out the J28  
glycotope unless Core2GlcNAc-T activity is present. Further transfection  
with FUT3 cDNA increased the antibody recognition. Nevertheless,  
transfection with FUT3 or FUT7 alone did not generate the formation of

the

J28 glycotope on the C-terminal peptide. Furthermore, the Cter peptide  
produced by CHO-K1 cells expressing Core2GlcNAc-T was more reactive to

the

mAbJ28 after in **vitro** fucosylation with the  
~~recombinant sol. form of FUT3.~~ These data suggested that the J28  
glycotope encompasses structures initiated by Core2GlcNAc-T and further  
fucosylated by .alpha.3/4-Fuc-T such as FUT3, likely on GlcNAc residues.

REFERENCE COUNT: 60 THERE ARE 60 CITED REFERENCES AVAILABLE FOR  
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L3 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 3

ACCESSION NUMBER: 1996:367198 CAPLUS

DOCUMENT NUMBER: 125:55468

TITLE: A missense mutation in the FUT6 gene results in total/  
absence of .alpha.3-fucosylation of human  
.alpha.1-acid glycoprotein

AUTHOR(S): Brinkman-Van der Linden, Els C. M.; Mollicone,  
Rosella; Oriol, Rafael; Larson, Goeran; Van den  
Eijnden, Dirk H.; Van Dijk, Willem

CORPORATE SOURCE: Dep. Med. Chem., Fac. Med., Vrije Univ., Amsterdam,  
1081 BT, Neth.

SOURCE: Journal of Biological Chemistry (1996), 271(24),  
14492-14495

PUBLISHER: American Society for Biochemistry and Molecular Biology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The major .alpha.3-fucosyltransferase activity in human plasma is encoded by the gene for fucosyltransferase VI (FUT6). A missense mutation (Gly-739 .fwdarw. Ala) in this gene is responsible for deficiency of enzyme activity in plasma. To exam. whether this fucosyltransferase is the sole enzyme responsible for the .alpha.3-fucosylation of serum glycoproteins in the liver, we studied the fucosylation of three glycoproteins in sera of individuals with or without inactivated FUT3 and/or FUT6 gene(s) but with a functional FUT5 gene. .alpha.1-Acid glycoprotein was used as the principal reporter protein for liver .alpha.3-fucosyltransferase activity, because of its high fucose content. In all individuals with the FUT6 missense mutation Gly-739 .fwdarw. Ala

in

double dose, no fucosylation of .alpha.1-acid glycoprotein was found. This .alpha.1-acid glycoprotein was not intrinsically resistant to fucosylation, since it was susceptible to in **vitro** **fucosylation** using an .alpha.3/4-fucosyltransferase isolated from human milk. The same result was found for .alpha.1-antichymotrypsin and .alpha.1-protease inhibitor. On the other hand in all individuals with .alpha.3-fucosyltransferase activity in plasma, .alpha.3-fucosylated glycoforms of the glycoproteins studied were found. The degree of fucosylation of .alpha.1-acid glycoprotein was correlated with .alpha.3-fucosyltransferase activity. These data indicate that the product of FUT6, but not of FUT3 or of FUT5, is responsible for the .alpha.3-fucosylation of glycoproteins produced in liver and suggest that this organ is a major source of .alpha.3-fucosyltransferase activity in plasma.

L3 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2002 ACS

DUPLICATE 4

ACCESSION NUMBER: 1997:45578 CAPLUS

DOCUMENT NUMBER: 126:128554

TITLE: Identification of a GDP-L-fucose:polypeptide  
**fucosyltransferase** and enzymic addition of  
O-linked fucose to EGF domains

AUTHOR(S): Wang, Yang; Lee, Geoffrey F.; Kelley, Robert F.;  
Spellman, Michael W.

CORPORATE SOURCE: Dep. Pharmacokinetics Metabol. Protein Eng., Genetech  
Inc., South San Francisco, CA, 94080, USA

SOURCE: Glycobiology (1996), 6(8), 837-842  
CODEN: GLYCE3; ISSN: 0959-6658

PUBLISHER: Oxford University Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB An assay for GDP-fucose:polypeptide **fucosyltransferase** has been established. The enzyme catalyzes the reaction that attaches fucose through an O-glycosidic linkage to a conserved serine or threonine residue

in EGF domains. The assay uses recombinant human factor VII EGF-1 domain as acceptor substrate and GDP-fucose as donor substrate. Synthetic peptides with sequences taken from five proteins previously shown to contain O-linked fucose (Harris and Spellman, 1993; Glycobiol. 3, 219-224)

did not serve as efficient acceptor substrates. These synthetic peptides did not comprise complete EGF domains and did not contain all six cysteine

residues that define the EGF structure. Therefore, the enzyme appears to require more than just a consensus primary sequence and likely requires that the EGF domain disulfide bonds be properly formed. The enzymic reaction showed linear dependency of its activity on time, amt. of enzyme,

and substrates. Although the enzyme did not exhibit an abs. requirement for Mn<sup>2+</sup>, enzymic activity did increase ten fold in the presence of 50 mM

MnCl<sub>2</sub>. The *in vitro* glycosylation reaction resulted in complete conversion of the acceptor substrate to glycosylated product, and characterization of the purified product by electrospray mass spectrometry revealed that one fucose was added onto the polypeptide. Most of the enzymic activity was found to be in the sol. fraction of CHO cell homogenates. However, when enzyme was prepd. from rat liver in the presence of protease inhibitors, 37% of the activity was recovered by Triton X-100 extn. of the membrane particles after extensive aq. washes. The result suggests that the enzyme is probably a membrane protein and,

by analogy with other glycosyl-transferase, probably has a 'stem' region that is very susceptible to proteolysis.

L3 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 5  
ACCESSION NUMBER: 1993:98970 CAPLUS  
DOCUMENT NUMBER: 118:98970  
TITLE: Glycosylation of rat sperm plasma membrane during epididymal maturation  
AUTHOR(S): Ram, Daulat; Tulsiani, P.; Skudlarek, Marjorie D.; Holland, Michael K.; Orgebin-Crist, Marie Claire  
CORPORATE SOURCE: Sch. Med., Vanderbilt Univ., Nashville, TN, 37232-2633, USA  
SOURCE: Biol. Reprod. (1993), 48(2), 417-28  
CODEN: BIREBV; ISSN: 0006-3363  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB The authors quantified 4 glycosyltransferase activities of spermatozoa and

fluid from various regions of the epididymis. Only 10-20% of the total glycosyltransferase activities (sialyltransferase, fucosyltransferase, galactosyltransferase, and N-acetyl glucosaminyltransferase) sedimented with the spermatozoa; the remaining 80-90% of were present in sol. form in

the epididymal fluid. When the 4 transferase activities were expressed per 10<sup>6</sup> spermatozoa, only sialyltransferase and fucosyltransferase activities showed maturation-dependent changes. The former enzyme was higher on the proximal caput spermatozoa and the latter on the distal caput spermatozoa. When spermatozoa from the proximal and distal caput, corpus, and proximal and distal cauda were incubated with fucose-labeled nucleotide sugar (GDP[<sup>14</sup>C]fucose), higher levels of radioactivity were routinely incorporated into the spermatozoa from the distal caput. The [<sup>14</sup>C]fucose-labeled spermatozoa or sperm plasma membranes, when solubilized, resolved in SDS-PAGE, and visualized by autoradiog., showed that the radioactivity had been incorporated into an endogenous acceptor of 86 kDa (major component) and several minor components. Treatment of the solubilized spermatozoa with N-glycanase suggested that the [<sup>14</sup>C]fucose is mainly present on N-linked oligosaccharide units. Thus, some of the sperm surface components are fucosylated during sperm maturation. The potential significance of the *in vitro* **fucosylation** of sperm surface components in the prodn. of functionally mature spermatozoa is discussed.

L3 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 6  
ACCESSION NUMBER: 1987:100089 CAPLUS  
DOCUMENT NUMBER: 106:100089  
TITLE: Increased fucosylation of chick brain proteins following training: effects of cycloheximide  
AUTHOR(S): McCabe, Norah; Rose, Steven P. R.  
CORPORATE SOURCE: Brain Res. Group, Open Univ., Milton Keynes, MK7 6AA, UK  
SOURCE: J. Neurochem. (1987), 48(2), 538-42  
CODEN: JONRA9; ISSN: 0022-3042  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
AB When chicks were trained to avoid pecking a bead coated with

methylantranilate in a 1-trial passive avoidance task there was an increase in fucose incorporation in vivo and in vitro in the right forebrain base of methylantranilate (M)-trained compared to water (W)-trained chicks. The relation of this increase to de novo protein synthesis in vivo and in vitro was examd. Cycloheximide (Cx), 1 mM, inhibited in **vitro fucosylation** of chick brain slices by 60% after 3 h. However, the training-related increase in in-**vitro fucosylation** still persisted. When Cx was injected intraventricularly 10 min before training, the subsequent increase in the **vitro fucosylation** due to training was still apparent. When Cx was injected and [14C]leucine and [3H]fucose incorporation studied in vivo in M-trained and W-trained chicks, there

was

no increase in fucosylation due to training in the Cx-treated M-trained over the W-trained chicks. Thus, in-**vitro fucosylation** and its increase subsequent to training is not protein synthesis-dependent, but both in vivo and in vitro there are interactions between Cx and fucosylation steps that are independent of Cx's effects on protein synthesis.

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END OF SEARCH HISTORY